# PROGRESS

OF

# MEDICAL SCIENCE.

## MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

REGIUS PROFESSOR OF MEDICINE, OXFORD UNIVERSITY, ENGLAND,

AND

W. S. THAYER, M.D.,

PROFESSOR OF CLINICAL MEDICINE, JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND.

**The Phagocytosis of Erythrocytes and the Question of Opsonins in Paroxysmal Hemoglobinuria.**—J. Eason (*Edin. Med. Jour.*, 1907, xxi, 440) states that most of the important work under discussion at present is on the determination of the identity of the substance in the blood which exercises the opsonic function and whether this differs from the amboceptor or immune body, or is like it. Barrett gave the name hemopsonin to that substance causing phagocytosis of erythrocytes. In paroxysmal hemoglobinuria hemolysis depends on the activity of an intermediary body or amboceptor whose activity is in abeyance if the blood is maintained (in vitro) at normal temperature. Under such circumstances no hemolysis occurs, but if such blood is cooled after being mixed with potassium oxalate to prevent coagulation, and then maintained at 37° C. in an incubator for three hours hemolysis follows. Control tubes of normal blood under similar conditions show no hemolysis. Active solution of the erythrocytes by phagocytes can be seen in the pathological blood after thirty minutes in ice-water and a similar period at 37° C. This process may be partly extracellular, as by contact with the leukocytes the erythrocyte is dissolved and the hemoglobin absorbed. "Phagolysis sometimes appears to be proceeding as rapidly as hemolysis, and the erythrocytes appear to undergo very rapid solution in the medium (complement?) thus set free from the leukocytes. This process may also be observed if the inactivated serum of a case of paroxysmal hemoglobinuria is mixed with washed corpuscles and leukocytes from a normal individual and is submitted to the conditions of temperature already described. This form of phagocytosis would, therefore, not appear to be dependent on the action of a thermolabile substance or normal opsonin, but on a thermostabile substance which may be either the hemolytic amboceptor or a body corresponding to the thermostabile opsonizing substance which may be artificially produced by immunizing an animal."

These observations may be made clinically by employing the method originally used by Ehrlich to simulate the condition of a paroxysm as follows: A ligature is put around the finger and then the finger put in ice-water (5 to 10 minutes) and then in warm water a similar length of time. Blood smears are then examined. Eason concludes as follows: "Thus it appears that phagocytosis is as much dependent on the previous chilling of the blood as is the extracellular lysis; another indication that the amboceptor is the cause of both processes."

**On the Behavior of the Eosinophile Leukocytes in Cases of Pulmonary Tuberculosis.**—JOHN M. SWAN and HOWARD T. KARSNER (*New York Med. Jour.*, 1907, lxxxv, 539). One of the authors (Swan) in 1904, in a study of the blood in 25 cases of pulmonary tuberculosis, came to the following conclusions: "The absence of the eosinophiles from the blood may be looked upon as an unfavorable prognostic sign. The increase of these cells while the patient is under treatment may be taken as an indication that the progress of the disease has a tendency to become arrested." This present study has to bear especially on this point and consists of the differential counts in 31 cases of pulmonary tuberculosis. In all cases 500 leukocytes were counted and the specimens were stained by Wright's method. The studies are from the Philadelphia General Hospital. In bedridden patients counts were made once a week or oftener if the case was ending fatally. In those not in bed, monthly counts were made. In a series of 31 cases, 26 were fatal and 5 were stationary or improved at the time the observations were discontinued. With the exception of 2 cases, the eosinphile cells were below 1 per cent. as a rule at periods varying from 102 days before death to the day of death. In these cases they varied from 1 to 2 per cent. for more or less lengthy periods before death. In the typically fatal cases the count was usually under 1 per cent.—as low as 0.2 per cent.; and the cells were even absent just before death. In the stationary or improved cases the eosinophilic count usually rose until discharge or discontinuance of the observations. The authors show interesting tables and charts and then append the counts of all cases in detail. They conclude as follows:

"In the light of these studies we should say that in cases of pulmonary tuberculosis the eosinophile cells tend to disappear from the circulating blood as the progress of the disease brings the fatal termination nearer, and that as the patient improves under treatment and as the disease shows a tendency to become arrested, the eosinophiles reappear in the circulating blood. It appears that this tendency of the eosinophile cells up and down may be of some use as a prognostic sign. It appears that this fluctuation of the eosinophile cells is a measure of the severity of the secondary infection with pyogenic organisms and not an indication of the extent of the tuberculous process itself; in fact the reappearance of eosinophilic cells in the circulating blood coincident with the improvement in the cases of phthisis is probably due to the removal of the chemotactic influence of the products of growth of the pus organisms and the reassertion of the influence of the tuberculin manufactured in the tuberculous lesions."